immediate identification and reduction of the CSF pressure by drainage may be beneficial.

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Discussion

Dr Lars G. Svensson *(Cleveland, Ohio).* I would like to thank the Association for the invitation to comment on the interesting and vexing problem of delayed deficits presented by Dr Safi in a large series of descending and thoracoabdominal aortic repairs. Unfortunately, they did not present the postoperative causes. We have seen it occur with hypotension from things like bleeding, arrhythmia, overdiuresis, sedation, reintubation, and other causes such as pulmonary emboli, arterial embolic showers, thrombosis of the intercostal, or lumbar artery patches or bypasses, and, in some patients, for no apparent reason.

There appear to be at least 3 common pathways: first, loss of spinal cord blood flow, particularly with a tenuous and perilous collateral blood supply; second, failure of adequate spinal cord oxygenation; and third, secondary spinal cord injury from the delayed complex biochemical cascade of deleterious pathways following ischemia and reperfusion.

The preoperative risk factors presented by Dr Safi of type II thoracoabdominal aneurysm, acute dissection, and renal disease reflect the problem of maintaining collateral blood supply in some of these patients. Because you only reattach intercostals from T8 to T12, according to your manuscript, do you think reattaching a wider range of intercostals or lumbar arteries may have reduced the risk? What are you now doing differently? We have found postoperative induced hypertension and delayed extubation useful. Do you think in patients with extensive aneurysms and few intercostal or lumbar artery ostia, endarterectomies should be performed for reattaching segmental arteries?

You had more deficits occur with CSF drainage. Do you think delayed CSF hypertension may be a factor? In a previous prospective randomized study in which postoperative hypotension resulted in 32% of the deficits, CSF drainage tended to be protective. In an excellent study recently published by Coselli, there also appeared to be some possible protection against delayed deficits. In 57% of your patients, delayed deficits improved with repeat drainage. Do you think that delayed removal of the catheter may be useful or do you think this would increase the risk of complications from the catheter too much?

Dr Safi. Thank you, Dr Svensson. With regard to our practice with intercostal arteries, we believe that intercostal arteries 8 to T12 are very important, and our work showed that these intercostal arteries are important for spinal cord perfusion. But in a paper from Amsterdam where they felt that if the lower intercostal arteries are not patent or occluded, the upper intercostal arteries are important, and currently, if we open the aneurysm and the intercostal arteries and the lower ones are not open, then we reattach the T5, 6, and 7. That is the only change.

With regard to the postoperative management, this is an intensivist study by Tam Huynh, and we currently believe that keeping a mean pressure of a 100 mm Hg is the order of the day, because hypotension correlates with paraplegia, as you and I have seen.

With regard to endarterectomy, I caution endarterectomy because I helped my late mentor, Dr Crawford, with 2 cases where the patient developed postoperative bleeding, one 2 hours after the endarterectomy and one 24 hours. I currently don't know how to reattach intercostal arteries to a graft, either an acutely dissected aorta or endarterectomy, because of risk of bleeding with a catastrophic event.

With regard to CSF drainage, we believe that CSF pressure, if it is high, may correlate with paraplegia. How long we are going to keep it, according to our median time for the CSF of the development of neurological deficits, is 2 days, so we are keeping it a day extra. I think if you keep it longer than that there is a risk of infection and some other complication.

Dr Ludwig von Segesser *(Lausanne, Switzerland).* I wish to congratulate Dr Safi for his excellent presentation, and I have a few questions with regard to the extent of repair. As a matter of fact, I did not see the exact proportion of thoracoabdominal repairs versus descending thoracic aortic repairs, and I believe that in acute aortic dissections, most of these cases can be repaired proximally first, and maybe there is a necessity for a distal reentry procedure.

I am asking this because in our environment there is an increasing proportion of endovascular aneurysm repairs, and under these circumstances there is no aortic crossclamping, and of course this may induce a shift or a selection bias in the patients you have referred.

Could you comment on this?

Dr Safi. In our experience, two thirds of our patients are extent I and extent II, so most of them are the more serious aneurysm. With regard to endovascular repair, there are reports in the literature that show delayed paraplegia following the insertion of the stent. With regard to acute dissection, if you remove the upper half of the descending thoracic aorta, risk of death, neurological deficit, or renal failure is minimal, but when we remove the thoracoabdominal aorta completely, from the left subclavian to the iliac bifurcation, the risk in our hands is about 35% to develop a neurologic deficit and the mortality rate is very high.